Glucagon-Like Peptide-1 Protects Human Islets against Cytokine-Mediated β-Cell Dysfunction and Death: A Proteomic Study of the Pathways Involved

Glucagon-like peptide-1 (GLP-1) has been shown to protect pancreatic β-cells against cytokine-induced dysfunction and destruction. The mechanisms through which GLP-1 exerts its effects are complex and still poorly understood. The aim of this study was to analyze the protein expression profiles of human islets of Langerhans treated with cytokines (IL-1β and IFN-γ) in the presence or absence of GLP-1 by 2D difference gel electrophoresis and subsequent protein interaction network analysis to understand the molecular pathways involved in GLP-1-mediated β-cell protection. Co-incubation of cytokine-treated human islets with GLP-1 resulted in a marked protection of β-cells against cytokine-induced apoptosis and significantly attenuated cytokine-mediated inhibition of glucose-stimulated insulin secretion. The cytoprotective effects of GLP-1 coincided with substantial alterations in the protein expression profile of cytokine-treated human islets, illustrating a counteracting effect on proteins from different functional classes such as actin cytoskeleton, chaperones, metabolic proteins, and islet regenerating proteins. In summary, GLP-1 alters in an integrated manner protein networks in cytokine-exposed human islets while protecting them against cytokine-mediated cell death and dysfunction. These data illustrate the beneficial effects of GLP-1 on human islets under immune attack, leading to a better understanding of the underlying mechanisms involved, a prerequisite for improving therapies for diabetic patients.

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